Parasitic Diseases

Learning Objective

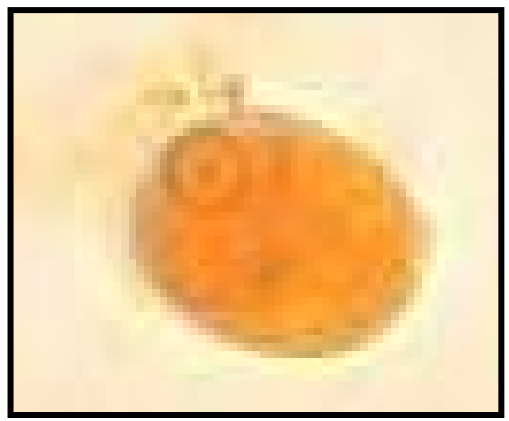
Describe the etiology, pathology, clinical features, diagnostic studies, management, and prevention of amebiasis, ascariasis, malaria, and toxoplasmosis.

Introduction

- **We will discuss four major parasitic diseases:**
  - **Amebiasis**
  - **Ascariasis**
  - **Malaria**
  - **Toxoplasmosis**

Amebiasis - Etiology

- ***Entamoeba histolytica***
- **Three stages:**
  - **Trophozoite (active amoeba)**
  - **Inactive cyst**
  - **Intermediate precyst**



E. histolytica Cyst

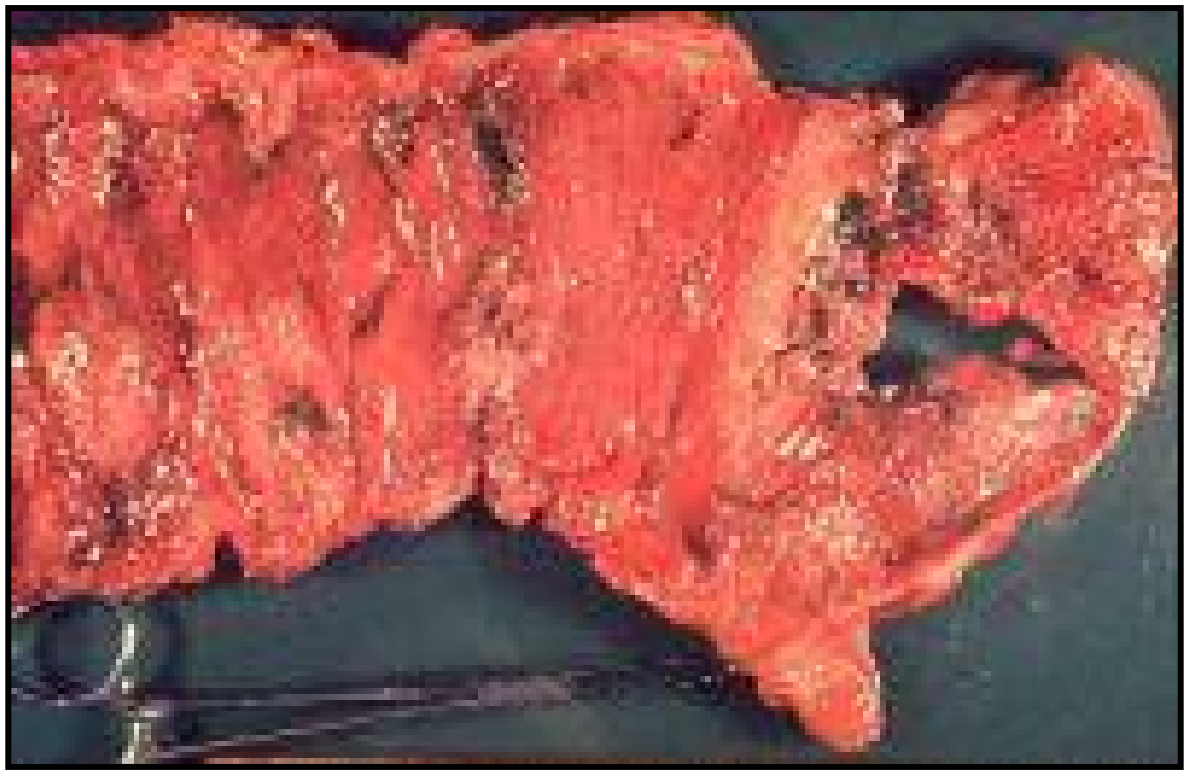
Amebiasis - Etiology

Free-living E. histolytica with Ingested RBCs

Amebiasis - Pathology

- **Dysentary acquired by ingestion of cysts found in contaminated food or water**
- **Often in those returning from tropics, individuals in institutions, and in homosexual males**
- **Excystation occurs in small intestine**
- **Trophozoites become established in lumen of large intestine**

Amebiasis - Pathology



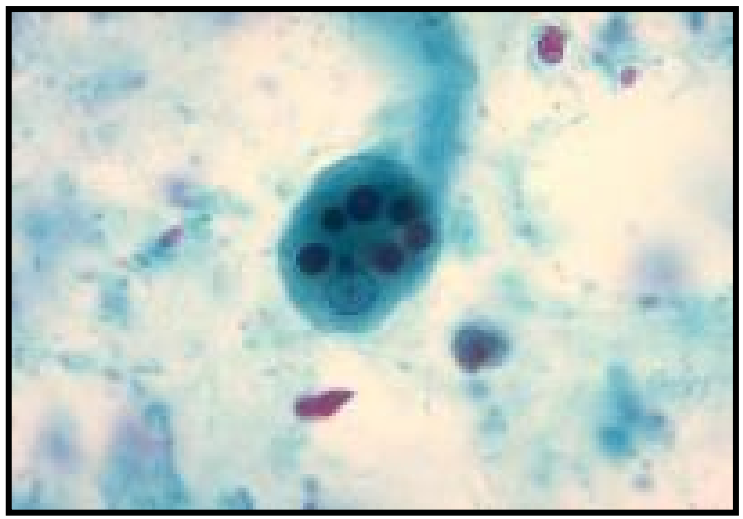
Amebiasis Intestinal Ulcer

Amebiasis – Pathology (cont’)

- **Trophozites invade intestinal epithelium and eventually penetrate venules and lymphatics**
- **Gain access to liver by way of portal vein**
- **Liver is primary extraintestinal site**

Amebiasis – Pathology (cont’)

- **Trophozoites may disseminate into lung, pleural cavity, pericardium through the bloodstream to brain**



Trophozoite

Amebiasis – Clinical Features

- **Following incubation period of 5 days to 2 weeks, symptoms of:**
  - **Diarrhea**
  - **Abdominal Cramps**
  - **Nausea**
  - **Vomiting**
  - **Tenesmus**

Amebiasis – Clinical Features (cont’)

- **Also possible:**
  - **Vague abdominal discomfort**
  - **General malaise**
  - **Loss of appetite**
  - **Weight loss**
  - **Mental apathy**
  - **Cutaneous lesions in perianal area**

Amebiasis – Clinical Features (cont’)

- Watery or formed feces containing mucus and blood
- Tender liver enlargement and colitis classic in amebic hepatitis
- Brain abscesses may progress to meningeal signs

Amebiasis – Diagnostic Studies

- **Other parasites look similar to E. histolytica**
- **Identifying E. histolytica in feces or tissues obtained from lesions**
- **Amebic serology using:**
  - **Enzyme-linked immunosorbent assay (ELISA)**

Amebiasis – Diagnostic Studies (cont’)

- **Indirect hemagglutination**
- **Agar gel diffusion**
- **Counterimmunoelectrophoresis positive in >90% of patients with invasive disease**

Amebiasis – Management

- **Choice of amebicides based on location and severity of infection**
- **Metronidazole for acute colitis or amebic liver abscess**
- **Iodoquinol, Paromomycin, or Diloxanide furoate for asymptomatic cyst passers**

Amebiasis – Prevention

- **Adequate disposal of human feces and water sanitation**
- **Proper handwashing after toilet use**

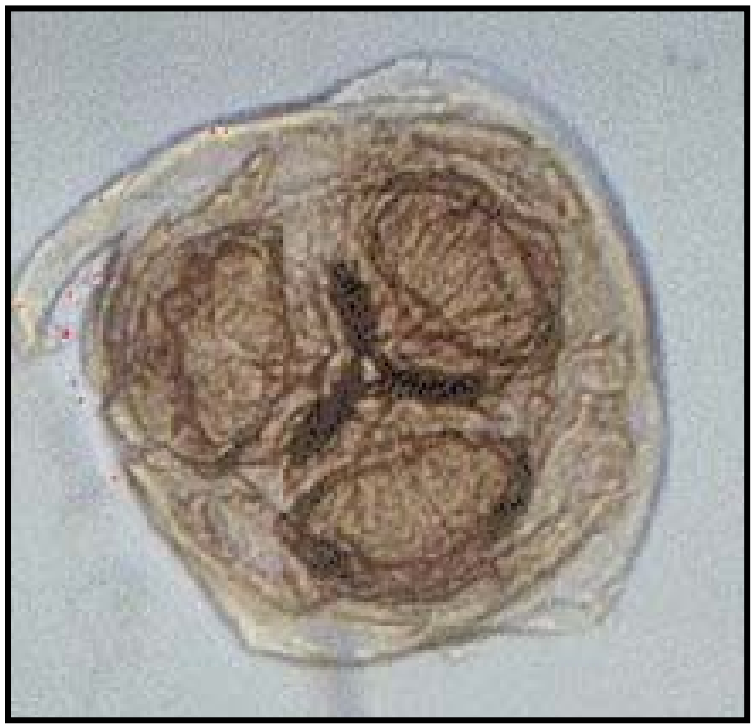
Travelers

- **Eat cooked food or self-peeled raw fruits and vegetables**
- **Drink bottled water**

Ascariasis - Etiology

- **Ascaria lumbricoides**
- **Most common human infection caused by worms**
- **Ascariasis is the largest and most common intestinal helminth**
- **Roundworms 6-13 inches in length; can be as thick as a pencil**
- **Can live1-2 years**

Ascariasis - Etiology



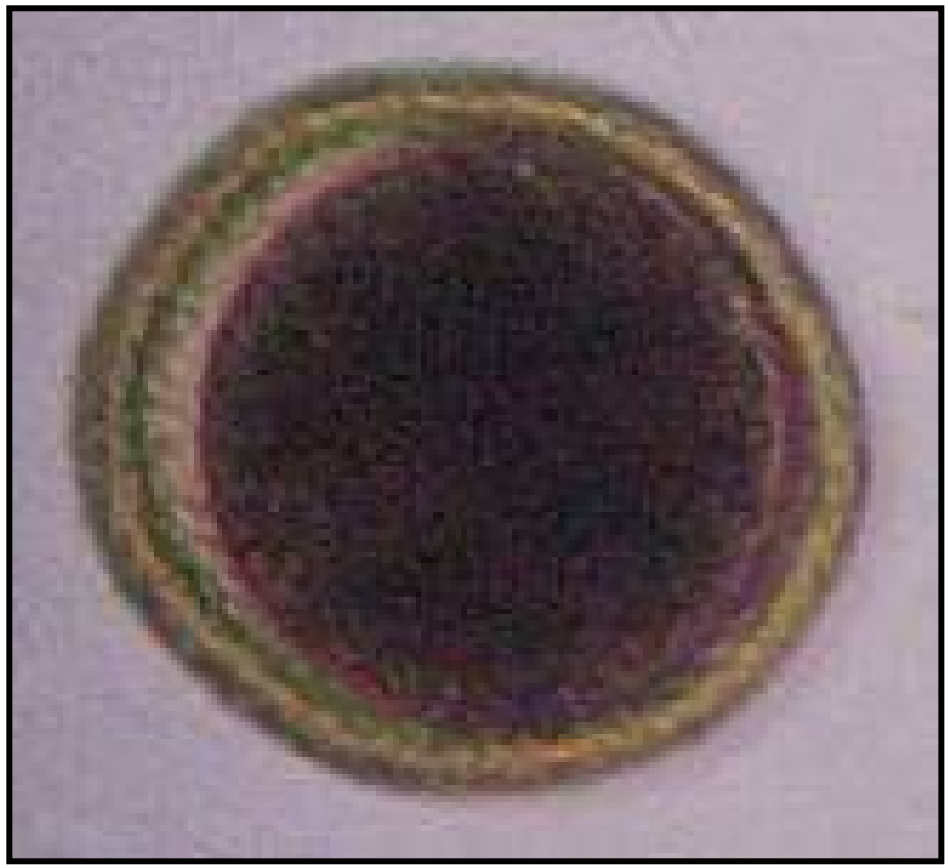
Roundworm

Roundworm from Colon

Ascariasis - Pathology

- **Adult ascarides inhabit the small intestine and deposit eggs into intestinal lumen**
- **Eventually passed in the feces**
- **Eggs mature in soil a minimum of three weeks before becoming infective by ingestion by the host**

Ascariasis - Pathology



Roundworm Egg

Ascariasis - Pathology

- **Larvae from infested eggs enter bloodstream, pass through alveoli, blocked at pulmonary capillaries**
- **Rupture into alveolar spaces, coughed up and subsequently swallowed and regain access to the intestines**
- **Children are most susceptible**

Ascariasis – Clinical Features

- In small worm load – may be asymptomatic
- In higher worm loads, may include:
  - Fever
  - Cough
  - Wheezing
  - Shortness of Breath

Ascariasis – Clinical Features (Cont')

- Intestinal blockage abdominal distention
- Biliary tract blockage
- Eosinophilia
- Oxygen desaturation
- Migratory pulmonary infiltrates
- Occasionally death from respiratory failure

Ascariasis – Diagnostic Studies

- **Stool specimen - Characteristic egg recovered in feces**

- **Pulmonary phase diagnosed by finding larvae and eosinophils in sputum**

Ascariasis – Management

- **Management**
  - **Pyrantel pamoate**
  - **Mebendazole**

Ascariasis – Prevention

- **Prevention**
  - **Proper disposal of human waste**
  - **Proper handwashing**
  - **Avoid opportunities to ingest contaminated food and water**
  - **Keep child from putting things in his/her mouth**

Malaria - Etiology

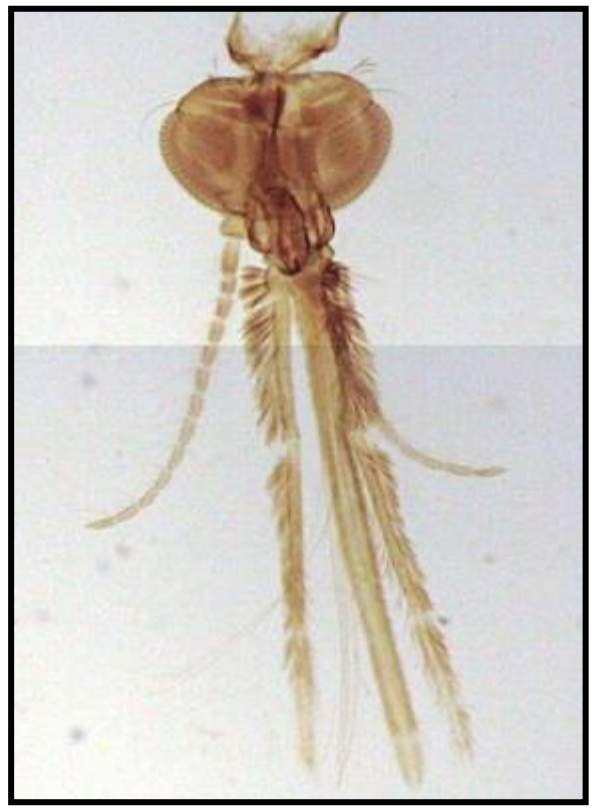
- **Known to infect humans:**
  - ***Plasmodium falciparum***
  - ***Plasmodium vivax***
  - ***Plasmodium ovale***
  - ***Plasmodium malariae***

Malaria – Etiology (cont’)

- **Occurs in more than 100 countries with 40% of the world population at risk**

Malaria – Etiology (cont’)

- **Derived from the bite of a malaria-infected Anopheles mosquito**
- ***P. falciparum* accounts for majority of infections and is most lethal**



Anopheles Mosquito

Malaria – Pathology

- **Sexual phase - in the Anopheles mosquito**
- **Mosquito then bites human and parasites are passed to human**
- **Asexual phase - in humans infected by mosquito bites (takes place in liver cells)**

Mosquito Biting Human

Malaria – Pathology (cont’)

- **Erythrocytic phase**
  - **Attachment to red blood cell surface**
  - **Progresses to intracellular parasite**
  - **Enlarges, divides producing multinucleated early schizont**

Malaria – Pathology (cont’)

- **Erythrocytes burst and free parasites**
- **Produces first clinical manifestation of the disease – Fever**
- **Invade other RBCs and repeat asexual cycle**

Malaria – Pathology (cont’)

- **Recurring asexual cycles involve enough erythrocytes until development of host immunity closes erythrocytic cycle**
- **Dormant hepatic sporozoites may resume intrahepatic multiplication leading to relapses**
- **Curable with prompt diagnosis and treatment**

Malaria – Clinical Features

- **Fever associated with red cell rupture**
- **Shivering**
- **Joint Pain**
- **Headache**
- **Lethargy**
- **Repeated vomiting**
- **Convulsions**
- **Coma**

Malaria – Clinical Features (cont’)

- Anemia (disproportionate to degree of parasitism)
- Jaundice
- Marrow function depression
- Spleen enlargement

Malaria – Clinical Features (cont’)

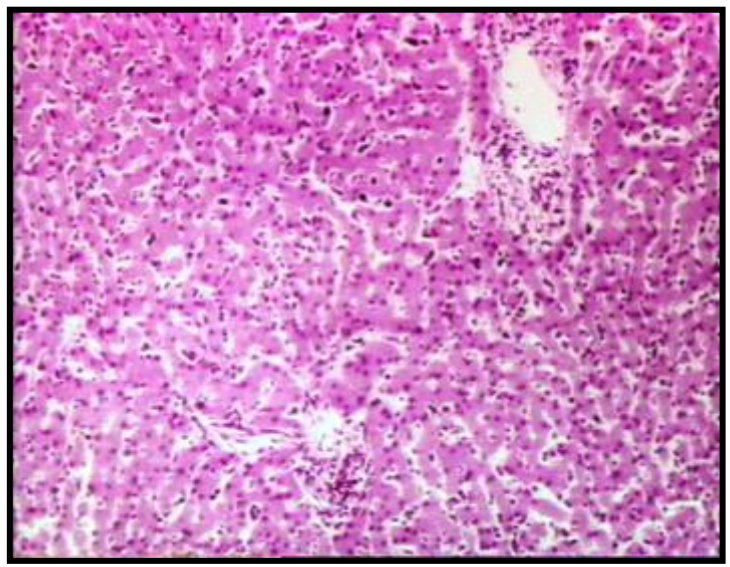
- **Hemolysis**
  - **If massive, can develop into hemoglobinuria resulting in dark urine**
- **Thrombocytopenia is common in malaria**

Malaria – Clinical Features (cont')

- **Vasodilatation due to high fever may lead to hypotension and decreased blood flow to vital organs**
- **May also see acute transient glomerulonephritis or progressive renal disease**

Malaria – Clinical Features (cont')

- ***Plasmodium falciparum*, if not promptly treated, may cause:**
  - **Kidney failure**
  - **Seizures**
  - **Mental confusion**
  - **Coma**
  - **Death**



P. falciparum

Malaria – Diagnostic Studies

- **Malarial parasites demonstrated in stained smears (thick or thin) of peripheral blood in virtually all symptomatic patients**

- **Serologic tests**

Malaria - Management

- **Treatment requires destruction of all forms of parasite**
- **Treat clinical attack of erythrocyte schizont with:**
  - **Chloroquine**
  - **Chloroquine-resistant falciparum malaria treated with quinine, antifolates or sulfonamides**

Malaria – Management (cont’)

- To prevent transmission, treat erythrocytic gametocyte with
  - Chloroquine (relapsing malaria)
  - Primaquine (falciparum malaria)

- To effect radical cure of hepatic schizont, treat with primaquine

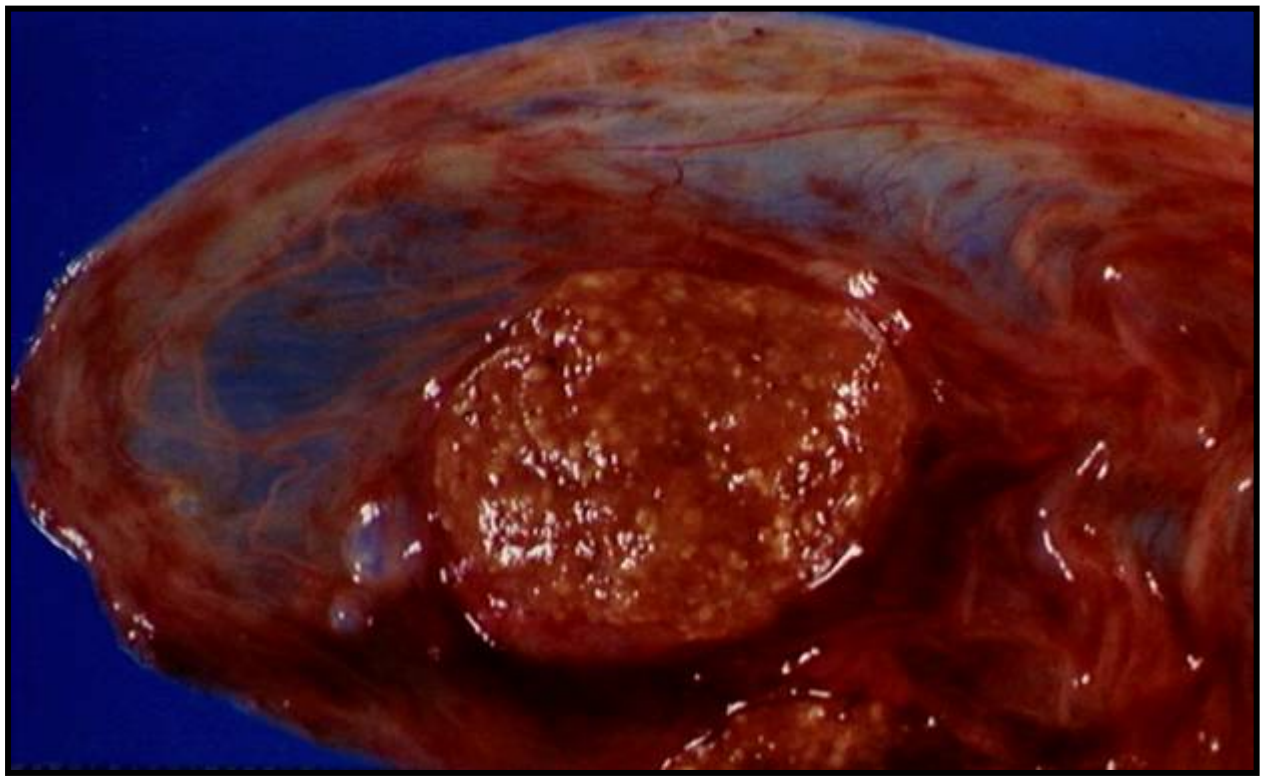
Malaria – Prevention

- **Avoid contact with mosquito vector**
  - **Remain indoors**
  - **Protective clothing**
  - **Repellent**
- **Anti-malarial drugs prior to travel to high-risk countries**

Toxoplamosis - Etiology

- **Toxoplasma gondii**
- **Accidental ingestion of contaminated cat feces**
- **Ingestion of raw or partly cooked meat**
- **Touching mouth after handling undercooked meat**

Toxoplamosis - Etiology



Placental Cotyledon Showing Toxoplasmo Gondii

Toxoplamosis – Etiology (cont')

- Contaminated utensils, cutting boards, and other foods after contact with raw meat
- Toxoplasma-contaminated drinking water
- Rarely, through infected organ transplant or blood transfusion

Toxoplasmosis – At Risk Populations

- Infants of mothers infected during or just before pregnancy
- Those with severely weakened immune systems
  - Acute or prior infection is reactivated and causes damage to brain, eyes, and other organs

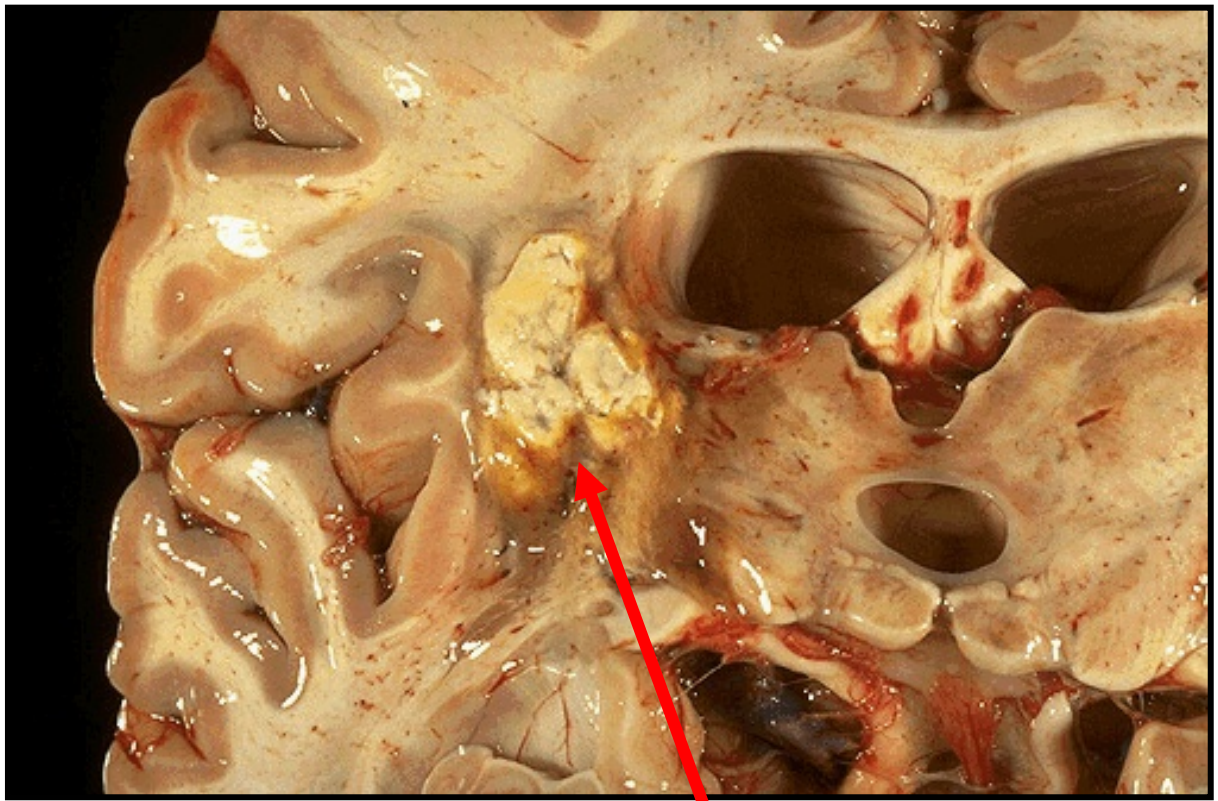
Toxoplamosis - Pathology

- **Parasite reproduces in the feline intestines and is shed in cat feces.**
- **Sporulation completed in external environment in 1-3 weeks**
- **Asexual cycle of macrophage rupture and release new parasites**
- **Many parasites destroyed as host develops immunity**

Toxoplamosis – Pathology (cont’)

Some trophozoites produce membrane forming cyst in organs such as brain, heart, skeletal muscle protecting multiplication and producing parasitologic relapse

Toxoplamosis – Pathology (cont')



Toxoplasmosis Cyst in the Brain

Toxoplamosis – Clinical Features

- **Normal / Immunocompetent Host**
  - **Asymptomatic localized lymphadenopathy frequently involving cervical nodes**
  - **Often accompanied by fever, fatigue, sore throat, rash, hepatosplenomegaly, atypical lymphocytosis**

Toxoplamosis – Clinical Features (cont')

- **Congenital toxoplasmosis**
  - **Newly-infected pregnant woman passes infection to the fetus**
  - **Common due to poorly developed immune mechanisms**
  - **Infection of CNS often catastrophic and results in abortion and still births**

Toxoplamosis – Clinical Features (cont’)

- **Congenital toxoplasmosis (cont’)**
  - **Live born children may have:**
    - **Microcephaly**
    - **Hydrocephaly**
    - **Convulsions**
    - **Psychomotor retardation**
    - **Fever**
    - **Hepatitis**
    - **Pneumonia**
    - **Skin rash**

Toxoplamosis – Clinical Features (cont')

- **Immunocompromised Host**
  - **Infection can be severe**
  - **Immunocompromise caused from AIDS, therapies, transplants, lymph disorders**
    - **Result in reactivation of preexisting latent infections**
    - **Often involves CNS**

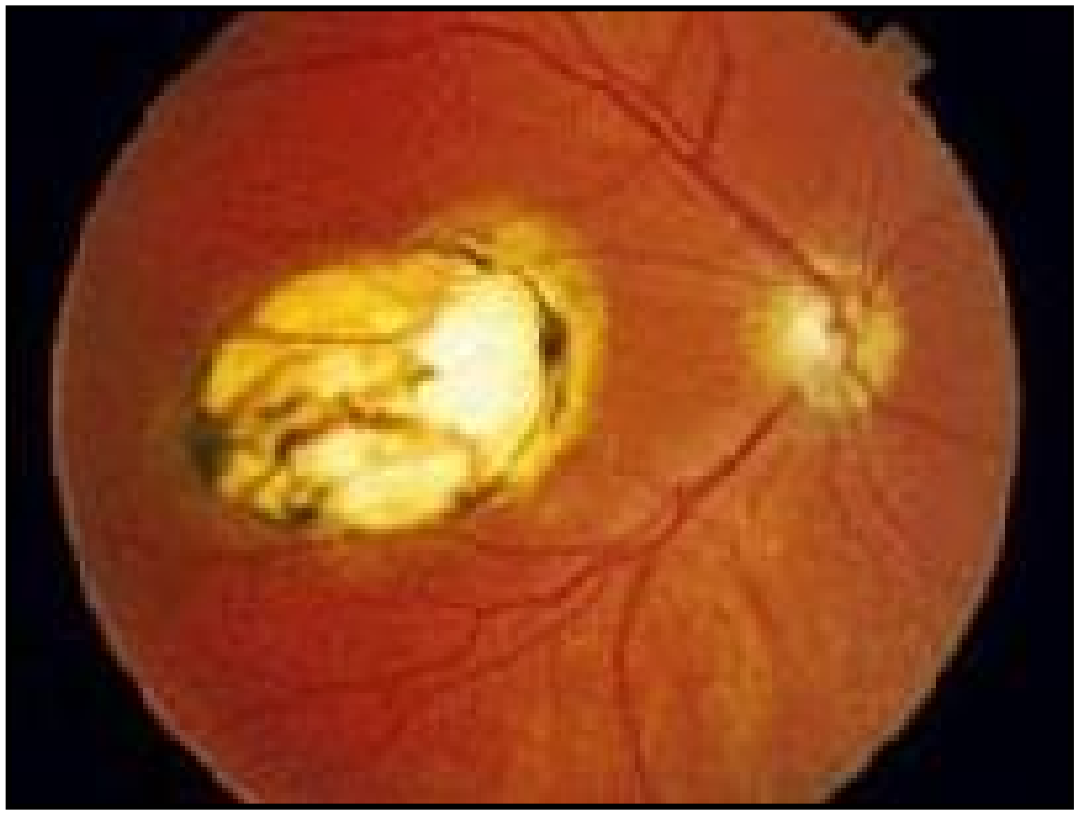
Toxoplamosis – Clinical Features (cont’)

- Immunocompromised Host (cont’)
  - Symptoms of meningoencephalitis or mass lesion
  - Demonstrates serious, often fatal wide-spread dissemination
    - Necrotizing pneumonitis
    - Myocarditis
    - Encephalitis

Toxoplamosis – Clinical Features (cont’)

- Congenital toxoplasmosis (cont’)
  - Those infected later in prenatal development demonstrate milder disease, but years later may develop:
    - Epilepsy
    - Retardation
    - Strabismus
    - Chorioretinitis

Toxoplamosis – Clinical Features (cont')



Ophthalmic Toxoplasmosis

Toxoplamosis – Diagnostic Studies

- **Serologic evidence of rising titers in IgG antibody between acute and convalescent serum specimens**
- **Detection of IgM antibodies provides more rapid confirmation of acute infection**
- **Demonstration of parasite in affected node**

Toxoplamosis – Management

- **Treat with Pyrimethamine plus Sulfadiazine or Trisulfapyrimidine for severe symptoms or when involvement of vital organs is present such as the eye**

Toxoplamosis – Management (cont’)

- **In immunocompromised and pregnant women treat acute infection with Pyrimethamine plus Sulfadiazine or triple sulfonamides or Clindamycin**

- **Pyrimethamine is teratogenic and contraindicated in first trimester of pregnancy**

Toxoplamosis – Prevention

- **Careful handwashing after handling uncooked meat and after outdoor activities**
- **Wash cooking utensils and cutting boards thoroughly**
- **Cook all meat thoroughly**
- **Peel and wash fruits and vegetables thoroughly**

Toxoplamosis – Prevention

- **Wear gloves when handling soil**
- **Avoid handling cat feces, particularly when changing cat litter**
  - **Use gloves and wash hands thoroughly**
  - **Change litter box daily**
  - **Do not adopt or handle stray cats**

Summary

- **Etiology, pathology, clinical features, diagnostic studies, management, and prevention of amebiasis, ascariasis, malaria, and toxoplasmosis**